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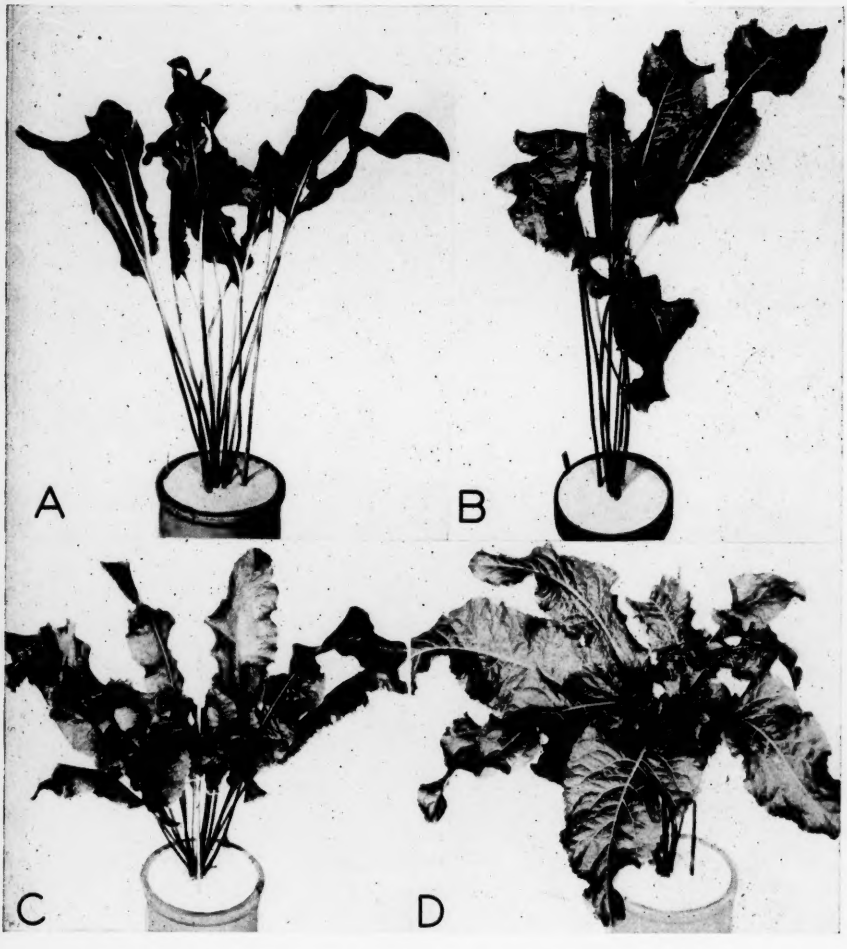

HORSERADISH SEEDLINGS—A BOTANICAL RARITY

(SEE PAGE 223)

VOLUME 40 AUGUST, 1949 NUMBER 8

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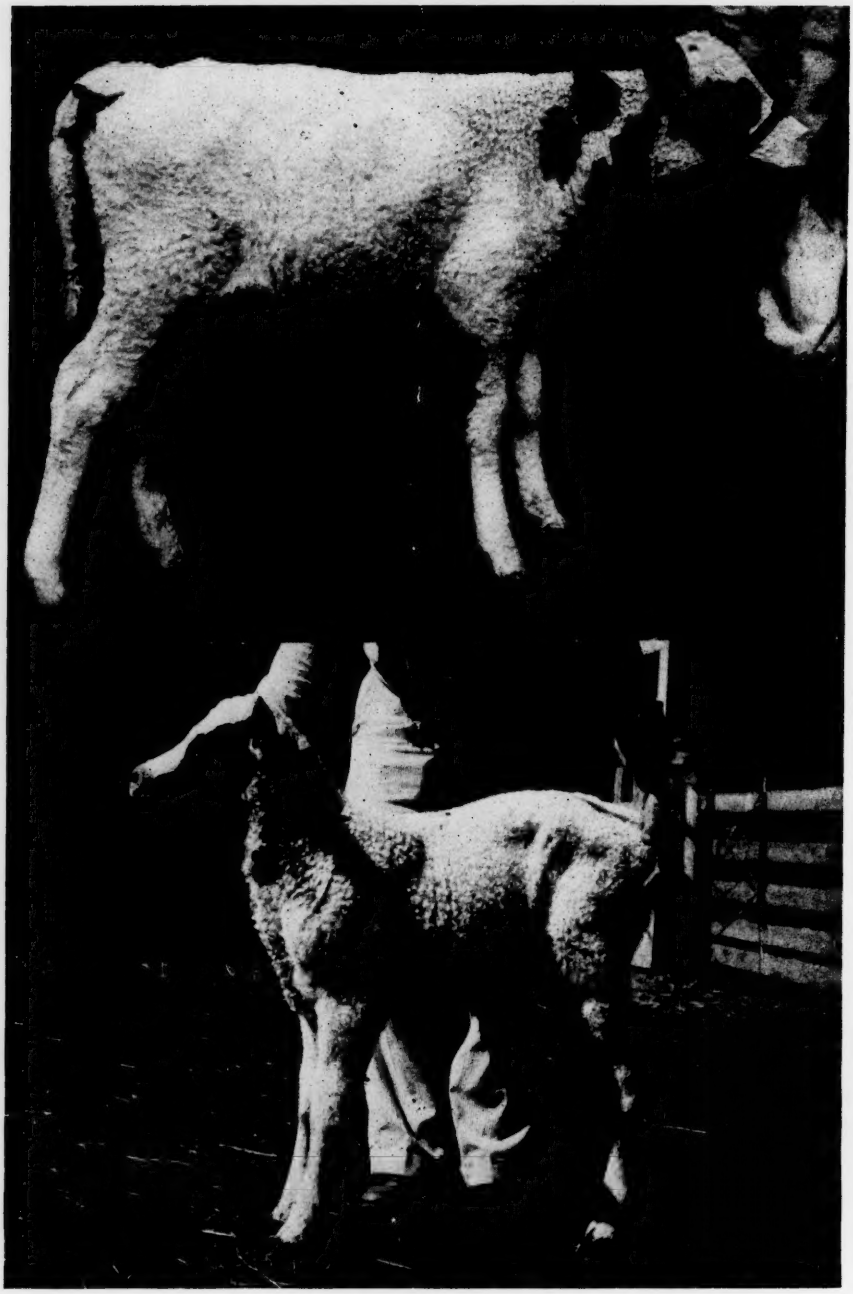


"FUR-BEARING CATTLE"

Figure 1

Two Ayrshire calves, six weeks of age, which were born with a "Karakul-type" curl in their coats. *A*—the bull calf, IV-4, in the pedigree chart (Figure 5). *B*—the first curly-haired daughter (V-3) of IV-4.

INHERITANCE OF A KARAKUL-TYPE CURLINESS IN THE HAIR OF AYRSHIRE CATTLE*

FRANKLIN E. ELDRIDGE, F. W. ATKESON, AND HEMAN L. IBSEN

AN unusual type of curliness of the hair in cattle was called to the attention of one of the authors in 1942 when a registered Ayrshire cow in which this characteristic was first noticed gave birth to a heifer calf with the same curliness. On February 10, 1945, the latter heifer gave birth to a curly-haired son which was purchased by Kansas State College with the objective of making enough matings to test the mode of inheritance of the characteristic. The last calf from these matings was born September 15, 1948.

Since this characteristic is not widespread in any of the breeds of cattle common to the U. S. A., it is fortunate that an accurate account has been obtained of the family of Ayrshires in which this apparent mutation occurred. With the mode of inheritance determined, breeders of cattle will be in a better position either to foster this trait or dispose of it. This is not the only occurrence of the Karakul-type of curliness in cattle. Johansson,[3] in 1942, reported the discovery of what seems to be the same genetic condition in the polled cattle of northern Sweden, and he called attention to an earlier report by Kronacher[4] that may have pertained to the same characteristic.

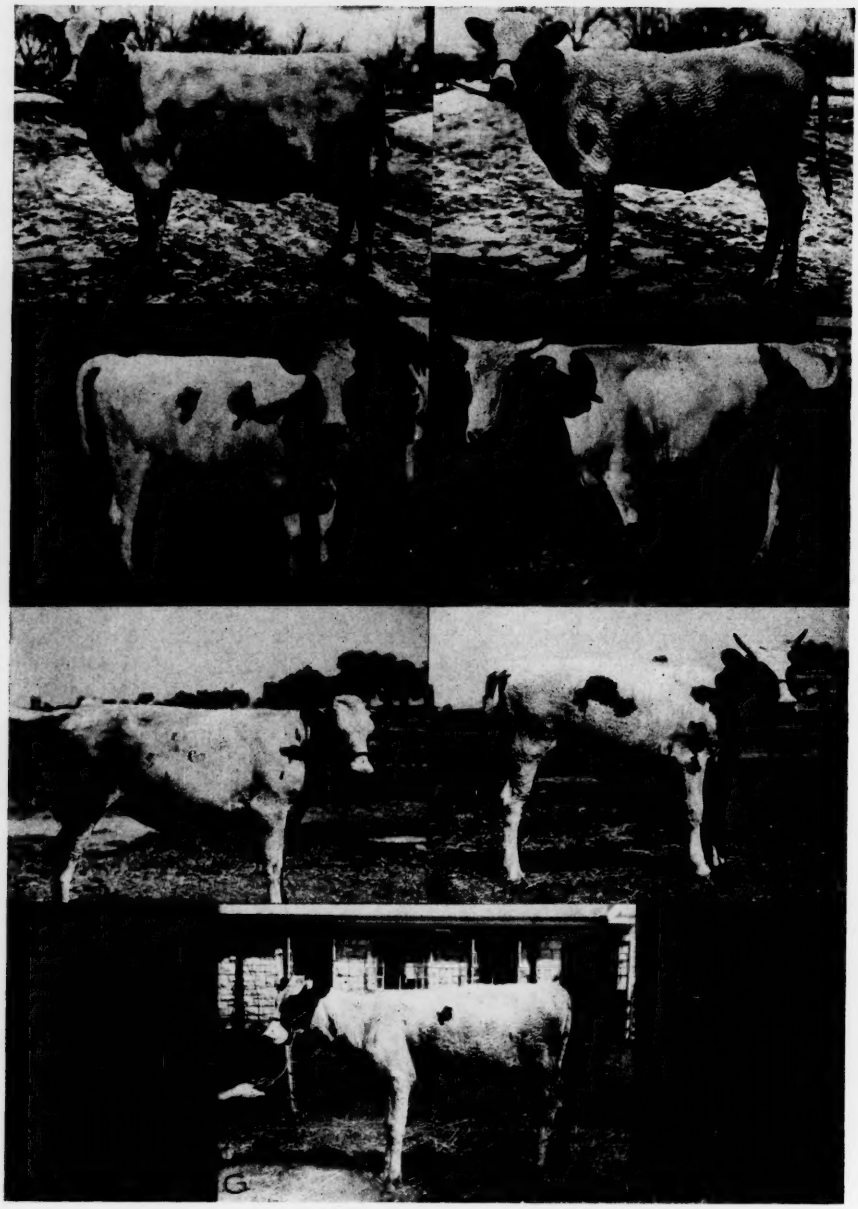
Description

At birth, the hair coat of the calves with curly hair closely resembles the coat of a newborn Karakul lamb. The hair is in rather small tight curls or ringlets over the entire body, tail, switch, legs down to the hocks and knees, and on the head except under the eyes and along the mouth and lips (Figure 1). Even the hair fringe on the ears is curly.

As the animal matures, the curly characteristic is less pronounced (Figures 1*A* and 1*B*; 2*B* and 2*E*), a change which is also typical of the Karakul breed of sheep. There is also a seasonal variation in the curly hair of the cattle, the curliness being much less evident in summer than in winter, probably due to the seasonal difference in length of hair (Figure 3). However, the hair on the poll and forehead, and to a certain extent the hair above the flank and on the thigh, remains curlier than in other regions of the body such as the side, neck and shoulder. This variation also is associated with differences in the length of hair. The least variable area of curliness seems to be in the hair forming the fringe on the ear since it retains the characteristic curliness at any age or season, whereas such hair is always straight on straight haired animals (Figure 6). As the curly-haired animal approaches the winter season, the hair grows longer over all parts of the body and the characteristic curl returns, so that even the mature animal has curly hair in most of the regions which are curly in calfhood, although possibly not so accentuated (Figures 2*A-B* and 3*A*). The casual observer might overlook the curliness in the summertime, particularly of the older animals, but it is easily identified during the winter months.

Two of the animals were recorded as slightly curly-haired at one time during the progress of this investigation and completely straight-haired at another time. The picture record which was kept on one of these animals (Figure 3)

*Contribution No. 184, Department of Dairy Husbandry, and No. 161, Department of Animal Husbandry, Kansas State College. The authors wish to acknowledge the initial microscopical work done by Carl Roubicek while working for his M.S. degree.



THE "CURLIES"

Figure 2

The above photographs show all the curly-haired animals involved in this investigation with the exception of III-5, which died soon after birth. The numbers (II-3, etc.) refer to the pedigree chart (Figure 5). *A*—the first known "curly," II-3; *B*—III-6, daughter of *A*; *C*—IV-3, daughter of *B*; *D*—IV-4, son of *B*; *E*, *F* and *G* are three daughters of *D*, numbered respectively V-3, V-4, and V-7.

provided the clue to this discrepancy. Although recorded as straight-haired soon after birth in June, this animal was classified as curly-haired the following April. When this animal was photographed the following August, it was classified again as straight-haired. In comparison, however, animals classified as curly-haired soon after birth were never later mistaken for straight-haired animals in spite of the seasonal variation mentioned above. Thus it was apparent that care must be exercised in the classification of animals at times other than at birth, or soon after; and furthermore, that single observations made by individuals who were not closely acquainted with the manifestations of this Karakul-type of curliness could not be relied upon to any great extent in determining its mode of inheritance.

In an effort to obtain a critical means for separating the straight-haired from the curly-haired animals, microscopic examinations of hair samples were made from both types.

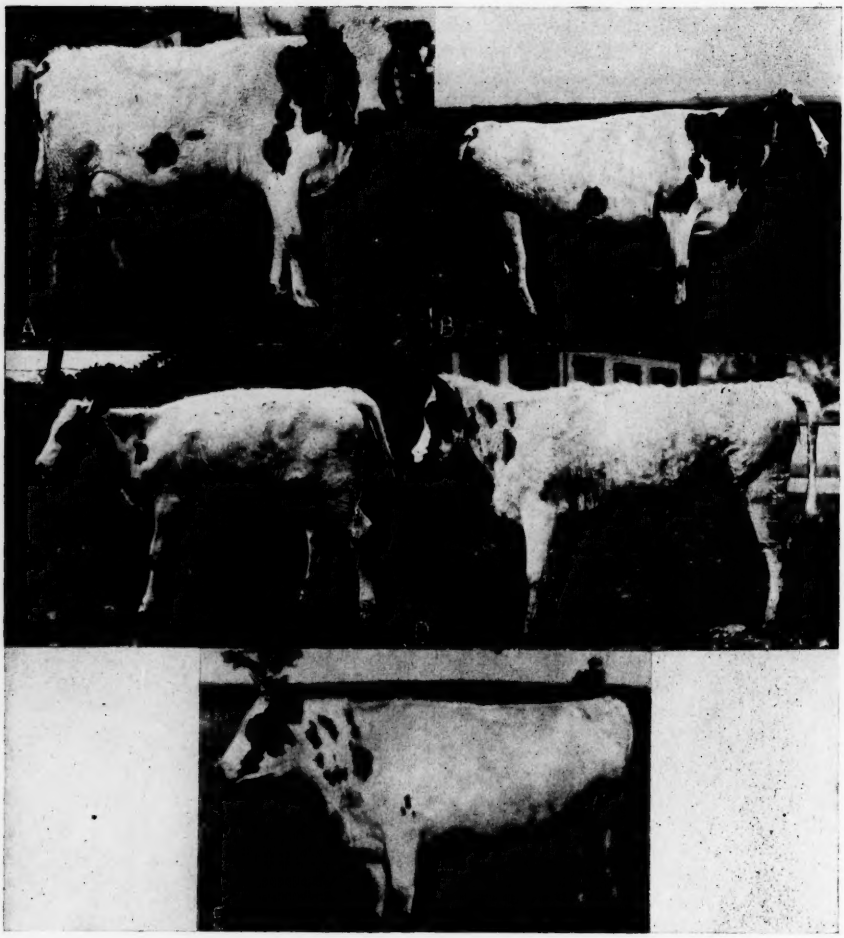
The cortex of a white bovine hair is transparent in contrast to the cortex of a red bovine hair, which contains many pigment granules. Consequently, when whole mounts of white hairs were used it was possible to examine in each hair the shape and general surface structure of the medulla as well as the over-all shape and structure of the entire hair. This fact was useful when comparisons were made between the medullae of hairs from straight-haired animals and the medullae of hairs from curly-haired animals.

Hair from the three curly-haired heifers, V-3,* V-4, and V-7 and hair from five of the straight-haired heifers, V-5, V-6, V-8, V-9, and V-10 were studied in cross-sections and whole mounts. The hair samples for examination were obtained on October 1, 1948, from the side of the body of each animal, in the region of the last rib. The straight-haired animals varied in age from two weeks to seven months, and the curly-haired animales varied in age from seven months to 14½ months.

A small subsample of hair from each animal was permanently mounted whole for microscopic examination. Another subsample from each animal was sectioned by a device developed by Hardy[2,8] before being mounted for observation. In order to facilitate comparison, specimens from a straight-haired animal were mounted on one end of each slide and specimens from a curly-haired animal were mounted on the other end. Observations were made with binocular microscopes at magnifications of 21× to 275×. Considerable detail was obtained by using both reflected light and transmitted light in the proper balance, especially at the higher powers of magnification.

The diameter of the hair from the curly-haired animals which was examined in whole mounts was found to be markedly irregular throughout its length in comparison with the very uniform, regular outer diameter of the hair from the straight-haired half sisters (Figure 4). The irregularly larger and smaller diameter of the medulla in the curly hairs was also quite evident, as it was observed in the white hairs. Furthermore, the medulla in the curly hairs had a more granular, rough appearance than the medulla of the straight hairs (Figure 4). The straight hairs were found to be quite cylindrical and uniform, with a relatively smooth surface of the medulla. Both types of hair tapered toward the distal end, with the medulla absent from the tip of the hair. In both types of hair there were some individual hairs which were lacking a medulla and some in which the medulla was discontinuous. Although the hair from the youngest, curly-haired heifer appeared flatter than any of the others examined, (Figure 4*A*), there was no consistent tendency for the hairs from the other two curly-haired heifers to be less cylindrical, or

*The symbols II-3, IV-1, etc., when used throughout this paper refer to the animals in the pedigree chart in Figure 5. The Roman numeral indicates the generation and the arabic numeral indicates the specific animal within that generation.



SEASONAL VARIATION IN DEGREE OF CURLINESS

Figure 3

Both straight-haired and curly-haired cattle vary in curliness, and occasional mistakes are possible in classifying adult animals. *A* and *B* are two photographs of Animal III-6 at different seasons: Early in the spring before shedding (*A*), the curliness extended over most parts of the body. In midsummer after shedding (*B*), the curliness was apparent only in restricted areas of the body. *C*-*E* show three photographs at different ages of the straight-haired, but rough coated animal IV-2, that at certain seasons might be confused with the Karakul-type of curliness. *C* shows IV-2 as a calf, and distinctly straight-haired. *D* is the same animal, as a calf in the early spring, questionably curly-haired. *E* shows the same animal in August, now distinctly straight-haired.

flatter than the hairs from their straight-haired half sisters. This observation is in contrast to the generally accepted opinion that curly hair is always flatter in cross-section than straight hair.[5,7,8] It has been recognized, however, that curliness of the hair is not invariably accompanied by flatness.[9]

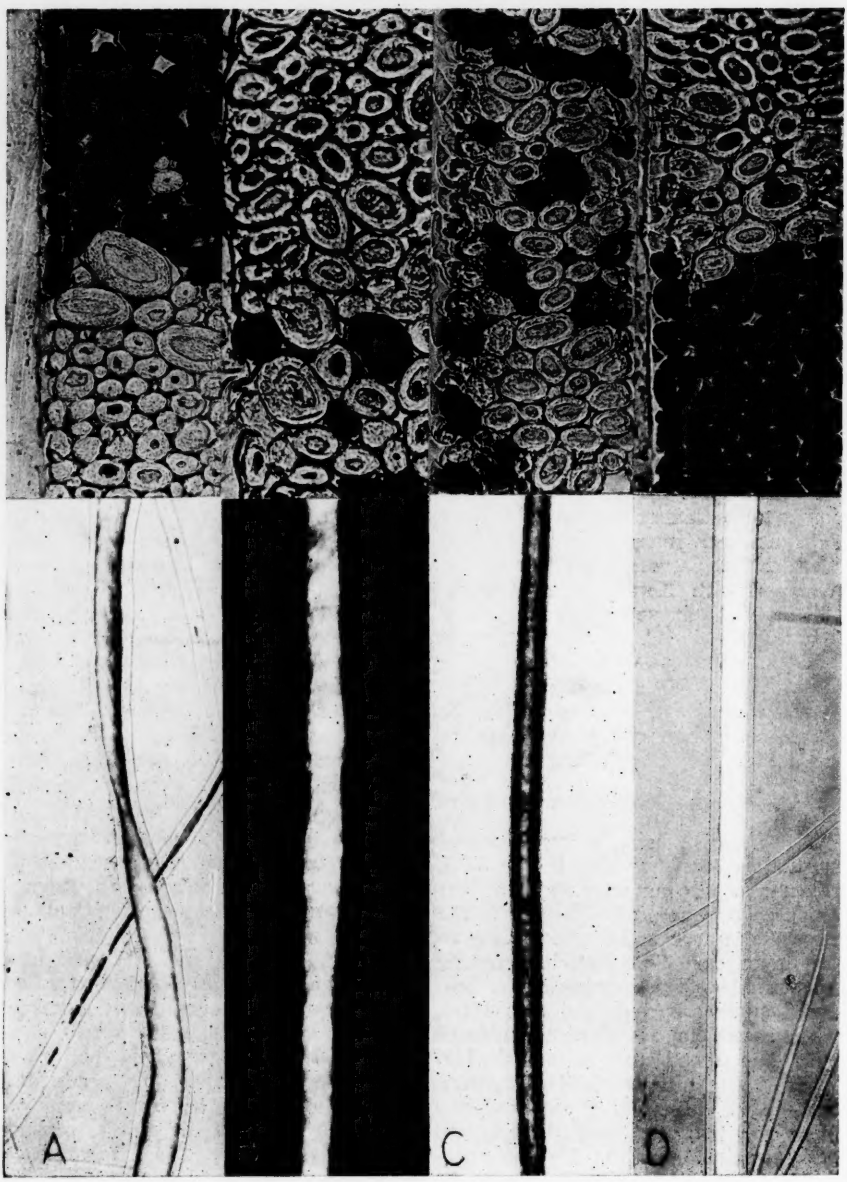
In the cross-sections (Figure 4) there were no consistent differences found between the straight hairs and the curly hairs. This is in agreement with Johansson[3] who also found no consistent differences in the samples he examined in cross-section from two ten-month old animals, one curly and one straight-haired. Nevertheless, the occurrence of an observable amount of flatness in the hair of the one animal (Figure 4*A*). similar to the flatness recored by Johansson[3] in a very young curly-haired calf, indicates that flatness in the hair can be combined with Karakul-type of curliness especially in young animals.

It was concluded, therefore, that the curliness transmitted by this specific gene was not due to the degree of flatness or roundness, but appeared to be due to the irregularity of the diameter of the hair and its medulla.

Mode of Inheritance

The first curly-haired animal observed by the authors was the female II-3. When mated to an unrelated bull, she produced a heifer calf, III-6, with the same appearance as herself, leading to the assumption that the characteristic was heritable. This same cow later produced another curly-haired calf, III-5, when mated to a different, unrelated bull. The second curly-haired calf, III-5, died soon after birth, but the first curly-haired heifer, III-6, produced four offspring, two curly-haired and two straight-haired. Each of the two curly-haired offspring, a male IV-4, and a female, IV-3, was sired by a different, straight-haired bull, unrelated to the dam. The two straight-haired offspring, a male IV-1, and a female, IV-2, were full sibs of the curly-haired female. Furthermore, three of the straight-haired offspring of II-3, half and full sibs of III-6, produced several offspring, none of which was reported to be curly-haired. These facts led to the hypothesis that a dominant gene controlled this characteristic. As a critical test of this hypothesis the curly-haired son of III-6, IV-4, was purchased by the Department of Dairy Husbandry of Kansas State College and was mated to ten registered Ayrshire cows, one registered Jersey cow and one grade Hereford. From these matings twelve heifer calves were produced, of which three were curly-haired and nine straight-haired. Tested by the Chi-square method this ratio of 9:3 was not a significant deviation from the 1:1 ratio which was to be expected on the hypothesis that a heterozygous male had been mated to twelve recessive females. Therefore, based on the observed ratio obtained from the above matings, and on the fact that in all cases where curliness of this type has been found it has been transmitted directly from parent to offspring, a valid assumption seems to be that the inheritance is due to a single dominant gene. The symbol *K* is suggested for this gene due to the phenotypic resemblance of the characteristic to curliness found in Karakul lambs.

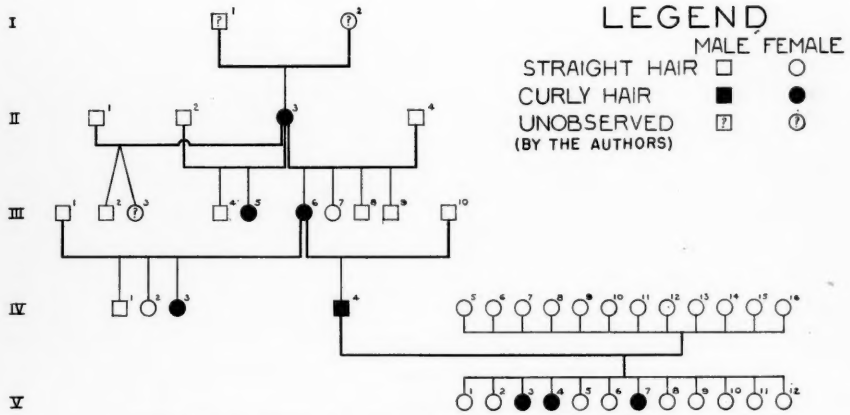
Although a single dominant gene is the simplest explanation for the mode of inheritance of this characteristic, alternative hypotheses must not be ignored, especially when such small numbers are involved. The observed 9:3 ratio is a perfect fit to the ratio of 3:1 expected on the following hypothesis: that Karakul-type of curliness is due to *two* non-linked, dominant genes, the bull IV-4 was heterozygous for both genes, and the twelve cows were double recessives. If such a hypothesis is correct, then the first curly-haired female, II-3, may have been simply the result of combining K_1 with K_2, two complementary dominant genes. Such an explanation would fit the breeding behavior of the curly-haired animals in the pedigree chart (Figure 5) even more closely than the simpler explanation. If further matings of curly-



CROSS-SECTIONS OF CURLY HAIR AND STRAIGHT HAIR

Figure 4

The top row shows cross-sections of samples of curly and of straight hairs (×120). The lower view shows "profile" microphotograph views of individual hairs. (The dark hairs are red and the light hairs are white.) The two left-hand samples (*A* and *B*) are of curly →



PEDIGREE CHART OF CURLY-HAIRED AYSHIRE CATTLE

Figure 5

The occurrence of curly hair in four successive generations, indicates that the expression of the Karakul-type curliness is conditioned either by one or two dominant autosomal genes.

haired animals continue to result in a marked scarcity of curly-haired offspring this alternative hypothesis may be proven to be the correct one. However, until such evidence has been accumulated the simpler hypothesis of a single dominant gene will be considered to be the more probable.

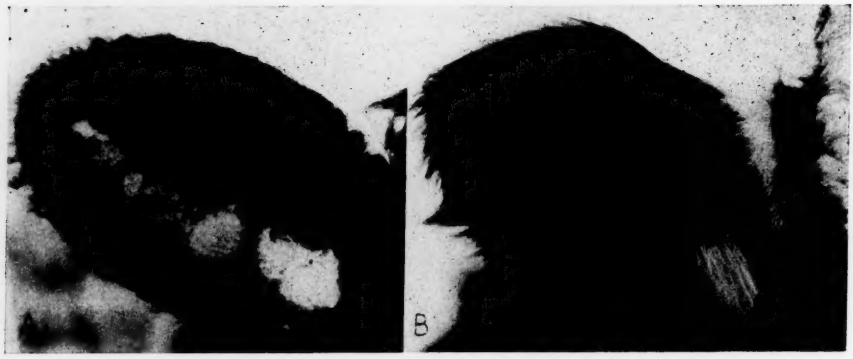
There is no indication from the information in the pedigree chart that Karakul-type of curliness is sex-linked since it has passed from female to female in two generations and from male to female and female to male in other cases. Consequently the gene is postulated to be autosomal.

Since up to the present time there have been no opportunities to mate two curly-haired animals, only heterozygous individuals have been observed. Thus, no information is available from which to draw any conclusions as to the degree of dominance of this gene. Furthermore, the lack of such observations makes it impossible to predict the appearance or condition of a homozygous dominant individual.

Origin of the Mutation

The fact that this characteristic Karakul-type of curliness in cattle has never come to the attention of the authors previous to 1942, and that it has been reported by only two other investigators[3,4] lends interest to the probable origin of the mutation reported herein. The extremely limited distribution of the characteristic must be due either to the recentness of the mutation or else to some type of selection against it. However, the very fact that the characteristic is not familiar to dairy cattle breeders is strong evidence that there has been no intentional selection, and from the breed-

hair. The two right-hand samples (*C* and *D*) are straight hair. No consistent differences between the straight and the curly hairs are revealed in the cross sections. The "profile" views (lower photographs) show that hairs from curly animals (*A, B*) are irregular in the diameter of the medulla and of the whole hair, accompanied by a definite flattening and twisting. The straight hairs (*C, D*) show a uniform regular diameter of the whole hair and of the medulla. All whole mounts were photographed using both reflected and transmitted light. The cross-sections and "profiles" of curly hairs are from animals V-7 (*A*) and V-3 (*B*). The straight-haired samples are from animal V-10 (cross section *C*) and V-6 (profile *C*) and from animal V-9 (*D*—both samples).



CURLINESS OF THE HAIR FORMING THE FRINGE ON THE EAR

Figure 6

This shows the contrast between the hair in the fringe of the ear of curly-haired (V-4) (*A*), and of straight-haired V-2 (*B*). The fringe-hair on the ears appears to be the most consistently distinct region for diagnostic purposes, of any part of the body.

ing behavior of the animals in this study there is apparently no natural selection against it. Consequently, it seems most probable to assume that the mutation is of recent origin.

Although the parents, I-1 and I-2, of the first curly-haired animal, II-3, were not observed by the authors, they were reported to have been straight-haired, the reports being considered quite reliable. If no mistake was made in the classification of these animals, mutation occurred in either the ovum or the spermatozoon which formed the zygote which became animal II-3. Thus, the cases reported by Johansson,[3] those reported by Kronacher[4] (if the latter was the same characteristic) and the ones reported herein probably are the result of a similar mutation which occurred in animals of different breeds, widely separated geographically.

Discussion

From the standpoint of inheritance, there would seem to be at least three different types of curliness in cattle. The three which are suggested are the Karakul-type of curliness, the curliness associated with semi-hairlessness,[1] and the more common variable type of curliness found distributed through most breeds of cattle.

Among animals observed at birth, there is an unquestionable distinction between those extremely curly-haired calves carrying the dominant, Karakul-type gene, *K*, and those possessing the recessive allele. However, at other ages and at different seasons, some confusion may occur.

The Karakul-type of curliness can be distinguished from the curliness associated with semi-hairlessness by the thickness of the coat, microscopic observation, and mode of inheritance. Craft and Blizzard[1] describe the semi-hairless characteristic as one in which the hair coat is extremely thin at birth and the hair is lacking entirely in some areas. At maturity the hair coat is thinner and the hairs are more wiry than in normal animals. In contrast, animals with the Karakul-type of curliness are at least as heavy-coated as the normal animals. Also, the curliness associated with semi-hairlessness increases with age to maturity, whereas the Karakul type of curliness is at its maximum at birth. Microscopically, the Karakul-type of curliness is distinguished by constrictions, or irregularity in diameter of the hair and its medulla, with no consistent tendency toward flattening. In comparison, Craft and Blizzard[1] state "microscopical observations . . . show that the

hairs have a tendency to be somewhat flat and spiral." and ". . . so far have not revealed constrictions or other abnormal appearances . . ." As a further distinction between these two types of curliness, semi-hairlessness is due to a simple recessive gene, whereas Karakul-type curliness is due apparently to a simple dominant gene.

The curliness found most commonly in cattle varies from slight waviness to distinct ringlets in the longer hairs. In mature animals, especially in the hot months when the body hair is short, this type of curliness can be confused easily with the Karakul-type of curliness. In both cases the curliness is most obvious in the regions of the poll and crest of the neck where the hair is longer. The Karakul-type may sometimes be distinguished by the greater degree of curliness found in the region of the flank and stifle joint, or in the tightness of the curl in the switch of the tail; but within the limits of observation of the authors the most reliable distinction between these two types is in the curl of the hair forming the fringe on the ears (Figure 6). Of a number of animals showing an extreme amount of the more common type of curliness observed by the authors, none had curly hairs in the ear fringe. However, among one herd of spring calves observed in November it was noted that the hair forming the fringe of the ear was inclined to be rather wavy in those animals whose hair coats were roughest. Thus, this distinction may not be perfect in some cases.

Curliness referred to in an informal report by Odlum[6] was apparently something entirely different from the characteristic reported herein. He described the animals as having "tightly curled hair" when born, but they were all males and the trait was in each case associated with a subsequent unthriftiness and swollen or stiff joints. Although one of the animals with Karakul-type curliness, III-5, was born with all four legs crooked and with the knee joints abnormally large, there was no reason to suspect this to be more than a coincidence since a straight-haired, full brother, III-4, also showed the same abnormalities, and since the other curly-haired animals in this study have all been normal anatomically and physiologically.

One of the authors found a registered Holstein-Friesian heifer in 1942 which was quite curly. Whole mounts and cross-sections of a hair sample obtained from this heifer were compared with those from the curly Ayrshires reported in this study and were found to be similar. The parents of the curly-haired Holstein were both reported to be straight-haired, but since the heifer was a non-breeder, no breeding tests were possible to determine the mode of inheritance. From the similiarity of the hair samples and comparison of a rather poor photograph with those of the curly-haired Ayrshires, it was assumed that the Holstein heifer also may have possessed the gene for Karakul-type curliness.

Two observations made during this study are receiving further attention. One is the fact that of the seven curly-haired animals, only one is a male, and the other is that this curly male, IV-4, has produced nothing but heifer calves in twelve matings. Both of these trends are based on a small number of individuals, but bull IV-4 is being used in additional matings and is being back-crossed to his three curly-haired daughters, V-3, V-4, and V-7 for further study.

Summary and Conclusions

1. A variation from the normal haircoat in cattle has been found in the Ayrshire breed. The hair is in tight curls on all parts of the body except the lower portions of the legs and around the muzzle, eyes and cheeks. In general appearance the hair coat is quite similar to the coat of newly born Karakul lambs.

2. The evidence indicates that this characteristic could be due to a single, autosomal, dominant gene, for which the symbol *K* has been suggested. Although the observed ratios fit more perfectly a hypothesis that the trait is due to two, complementary, dominant genes, the sim-

pler explanation is considered more probable.

3. This type of curliness differs from the more common type of curly or wavy hair of cattle in that the curliness in the calves is extreme over all parts of the body instead of mostly on the head and neck; it is equally pronounced in both sexes; and it is more uniform in its expression.

4. Further breeding work is being conducted in order to obtain, if possible, a homozygous dominant individual, and to obtain further proof concerning the mode of inheritance.

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✦

PREGNANCY DIAGNOSIS TESTS

Dr. Cowie's book* is a clear, orderly, compact review which demonstrates the world-wide interest in tests for pregnancy diagnosis. His impressive bibliography of around 2,000 publications covers the outstanding work in this field during the past quarter of a century. A brief historical review points out that the Egyptians were interested in this subject 3,000 to 4,000 years ago and suggests that certain tests may be thousands of years old.

Many of the various tests were first used in diagnosis of the pregnancy of women. In order to give a complete picture the reviews deal with human pregnancy and with pregnancy in many types of animals. These tests are based on seven different kinds of data:

(1) clinical methods; (2) hormonal investigations of body flunds; (3) enzymic investigations of body fluids; (4) other biochemical investigations of body fluids; (5) physiological phenomena; (6) immunological phenomena; (7) physical investigations of body fluids.

In discussing each of these, there is a summary of the outstanding work with adequate data on methods and the accuracy of the results observed to date in the types of individuals investigated. Tests have been run on about thirty species of animals such as the mare, cow, ewe, goat, sow, bitch, cat, rabbit, rat, mouse, guinea pig, ferret, deer, elephant, giraffe, ape, etc. Furthermore, the tests based on gonadotrophic hormones of pregnant women, mares, cows, etc., show the usefulness of a wide variety of test animals. The list of living organisms that have been exposed to pregnancy urine includes not only male and female mammals such as mice, rats, rabbits, guinea pigs and hamsters but also birds, amphibia, fish, plants and unicellular organisms.

Altogether the information based on hormonal investigations of body fluids runs through four chapters. In devoting this space to gonadotrophic, estrogenic and several other hormones, the field has been thoroughly covered.

Some of the physiological tests have shown considerable accuracy, but many of them give conflicting or undesirable results.

The enzymic and other biochemical tests are proving to be valuable in diagnosing human pregnancy.

The section on immunological phenomena is on allergic tests in women and female animals. These are divided into skin, eye and "haemoclastic" tests. Chorionic-gonadotrophin, placental-extract, colostrum, etc., have been injected either intradermally or subcutaneously into pregnant and non-pregnant females. The data indicate that these immunological tests have not been of sufficient accuracy to be of real value in pregnancy diagnosis.

The physical investigations of body fluids center around changes in the properties of blood, milk, urine and secretions from the cervix and vagina. Studies of surface tension, osmotic pressure, crystal configuration, mitogenetic radiation, etc., have been followed with conflicting results. Further examinations may show some of these methods to be useful as pregnancy tests. (*Continued on page* 218)

*Pregnancy Diagnosis Tests, Joint Publication #13. Commonwealth Agricultural Bureau, Wales. September, 1948.

A PEDIGREE OF PARTIAL ANODONTIA

W. G. Erwin and Robert W. Corkern*

PARTIAL anodontia in the pedigree here described involves the absence of the second bicuspid teeth and the third molars. Other permanent teeth are usually missing, but the various affected persons do not all lack the same teeth. So far as can be determined, the deciduous teeth are normal in number and arrangement. Where deciduous teeth are not replaced they usually are not shed, but persist along with the permanent teeth.

Pedigrees involving the partial absence of teeth are numerous in the literature. Reduced or absent upper lateral incisors are perhaps most common. Complete absence of incisors, lack of upper permanent cuspids, absence of third molars, and missing first and second bicuspids have been reported in a number of separate pedigrees. In most cases, the anomalies are inherited as simple dominants. Such an extensive summary of studies of hereditary tooth abnormalities is given by Gates[3] that further reference to the literature seems unnecessary here.

The Pedigree

Five members of the pedigree, II-12 and III-2, -4, -5, and -10 were examined clinically, and full mouth X-rays were secured of III-2 and -4. Information regarding the other individuals of the pedigree was obtained from I-2, II-2 and -12, and III-2.

At the age of twenty-eight I-1 consulted a dentist, who expressed great surprise at finding that he still had some of his deciduous teeth. Since he later became the father and grandfather of known affected individuals and since he retained deciduous teeth as late as the age of twenty-eight, exhibiting a tendency characteristic of all of the known affected persons, the authors believe that he can be classified as possessing the trait. I-2 was certain that she had a complete set of permanent teeth, and that this trait had never occurred on her side of the family.

At the age of nineteen II-2 consulted a dentist regarding the poor condition of his teeth. He was informed that his upper lateral and central, and lower lateral incisors were deciduous. Whether or not he retained other deciduous teeth was not ascertained. He had no third molars. At the age of twenty II-3 was informed by her dentist that her upper lateral incisors and cuspids were deciduous. These were removed and replaced by bridgework. II-5 and -10 had normal permanent dentition, including third molars, and never required any dental work. According to his mother, II-7 retained his deciduous lateral incisors and cuspids in the upper jaw and all four deciduous incisors in the lower jaw as late as the age of nineteen.

A clinical examination of II-12 revealed the absence of the second bicuspids and third molars in both jaws. The upper left, first molar had been extracted. Otherwise the permanent dentition was normal. III-1, who was twenty-three years old, was not available for clinical examination; hence the memory of immediate relatives had to be relied upon. Her only permanent incisors were the upper centrals. She also had no third molars.

Table I summarizes the data for four of the five members of the family who were examined clinically. III-2 had the fewest permanent teeth. Eight were missing above and ten below. The central incisors in the upper jaw were the only permanent incisors. Deciduous and permanent cuspids were present in the upper right jaw, the former being mesial to the latter. In the upper left

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jaw the permanent cuspid was somewhat intermediate between its usual position and that normally occupied by the lateral incisor. This condition resulted in conspicuous diastemas between the anterior teeth. There were no second bicuspids in either jaw and no first bicuspids in the upper jaw. The third molars were missing above and below. Full-mouth X-rays revealed no unerupted teeth in either jaw.

III-3 had a full set of permanent teeth, including the third molars which had either erupted or were partially erupted.

III-4, who was sixteen years old, should normally have all of her permanent teeth except the third molars. The central incisors in the upper jaw and the lateral incisors in the lower jaw were permanent. The other incisors were deciduous. The general appearance of the upper front teeth of III-2, -4, and -5 was almost identical, with broad central incisors, cuspids in the place of lateral incisors, and marked diastemas between them. III-4 had no second bicuspids, the spaces for them being occupied by the deciduous second molars. All of the permanent first molars were present, but only the lower right second molar had erupted. X-ray photographs showed the other three second molars in the process of formation, with crowns but as yet no roots. They were about as far advanced in development as normally occurs at seven to eight years of age. No third molars were evident in the X-rays.

III-5 was thirteen years of age at the time the data were collected. Normally he should have erupted all of his permanent teeth except the second and third molars. The second molars should be at least in the process of eruption. All permanent incisors were present except the upper laterals. The upper right first bicuspid was missing. The deciduous second molars were still in place in both jaws; so there were, of course, no second bicuspids. The four first molars were erupted, but the only permanent second molar was in the lower left jaw. Age precludes third molars.

III-10 was labeled as questionable regarding the tooth condition. At her age of nine years, she would be expected to have the following permanent teeth: first molars and central and lateral incisors. Of these she had the central incisors and first molars above, and the central and lateral incisors and first molars below. If she is to have normal permanent dentition, eruption of the upper lateral incisors should occur soon. Since no X-rays were made in her case, she must remain questionable regarding this trait.

Discussion

It will be noted from Table I that the second bicuspids and third molars were missing in all four individuals. Full mouth X-rays of III-2 and -4 failed to reveal even a trace of these teeth within

TABLE II.—Tooth Deficiency Tested for Linkage with Hair Color

				Tooth Deficiency	
			Hair	Similar	Dissimilar
No. Pairs	Chi-Square*	P	Similar	5	8
25	1.00	>0.30	Dissimilar	8	4

*Yates' correction for continuity has been applied.

TABLE I.*—Dentition of Four of the Persons Examined Clinically

		Upper Jaw												
		Right							Left					
Individual	Age	M-2	M-1	B-2	B-1	C	L.I.	C.I.	C.I.	L.I.	C	B-1	B-2	M-1	M-2
II-12	36	P	P	D	P	P	P	P	P	P	P	P	D-Ex.	P-Ex.	P
III-2	21	P	P	D	D	D&P	M	P	P	M	P	D-Ex.	D	P	P
III-4	16	M	P	D	P	P	M	P	P	M	P	P	D	P	M
III-5	13	M	P	D	D-Ex.	P	M	P	P	M	P	P	D	P	M
		Lower Jaw													
II-12	36	P	P	M	P	P	P	P	P	P	P	P	M	P	P
III-2	21	P	P	D-Ex.	P	D	D	D-Ex.	D	D	D	P	D-Ex.	P	P
III-4	16	P	P	D	P	P	P	D-Ex.	D	P	P	P	D	P	M
III-5	13	M	P	D	P	P	P	P	P	P	P	P	D	P	P

*Third molars are not shown in this table, since they were missing in all of the individuals. R, right; L, left; P, permanent; D, deciduous; M, missing permanent; ex., extracted.

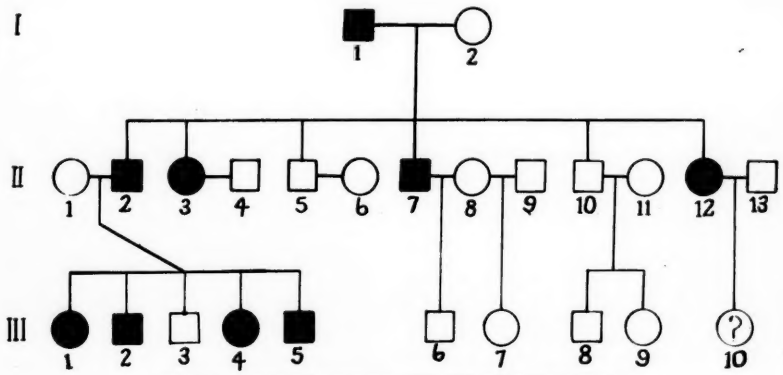

Figure 7

In this family varying numbers of missing teeth are found in nine individuals. Where the permanent tooth buds are lacking, there is a tendency for the juvenile teeth to persist. Simple dominance with variable penetrance accounts for the observed distribution. No linkage with hair color could be estabished.

the upper or lower jaws. Of course III-5 was too young to have erupted third molars, but since his dental pattern was otherwise comparable to that of the other affected individuals in the pedigree, it is assumed that he also lacked these teeth.

II-12 was the only one of the four examined clinically who had only eight missing permanent teeth. Thus this trait in its minimal effect eliminates the second bicuspids and the third molars. Otherwise, its expression is quite variable. Teeth in the upper jaw which were consistently unaffected were the central incisors, the cuspids, and first and second molars. III-4 and -5 had not erupted second molars, but X-rays showed them in the process of formation in III-4. It may be assumed that they will eventually erupt in both individuals. The lateral incisors and first bicuspids in the upper jaw were present in some individuals, absent in others.

In the lower jaw only the first bicuspids and the first and second molars were consistently present, assuming that III-5 will erupt the missing second molar. The lower, left, second molar had not been erupted in III-4, but X-rays revealed it in the process of formation. The central and lateral incisors and cuspids in the lower jaw were present in some cases and absent in others.

Besides the elimination of the second bicuspids and third molars, and the variability of expression of certain other teeth, a delay in the eruption of still others seems to be involved in this dental anomaly. This observation is supported by the fact that both of the second molars above and the left second molar below were present in the jaw but unerupted in III-4, who was sixteen years old. These teeth normally come through by the twelfth or thirteenth years.

Beadle[1] reported a pedigree in which missing teeth were inherited as a dominant. Fifteen affected individuals had dark hair and three had light hair, suggesting linkage. Burks[2] has shown further evidence of linkage between tooth deficiency and hair color. Burks' method of checking possible linkage was used in this pedigree. Hair color was appraised by matching the hair on the back of the head, close to the scalp, against the "Rapidol" chart of human hair color. Members of each sibship in

generations II and III were paired in all possible combinations within each sibship. The pairs so obtained were arranged in four classes according to the pairs that were similar or dissimilar for each of the two traits under consideration. The results of these calculations are shown in Table II. The *P*-value is slightly in excess of .30, which is extremely poor evidence for linkage. However, the number of pairs involved in this pedigree is too small to have significance. Also, it should be noted that Burks' data do not show conclusive evidence for linkage.

Burks advances a hypothesis that missing third molars is due to a simplex condition *Aa*, and that more extensive deficiencies are due to a duplex condition *AA*. Such a hypothesis would not explain the inheritance of tooth deficiency in the pedigree presented here, since all of the affected individuals have missing third molars plus additional deficiencies, and none of the individuals who married into the family had missing teeth.

In this pedigree the absence of third molars and second bicuspids may be adequately explained by simple dominance. The variability of additional missing teeth might possibly be due to differences in penetrance.

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Grants for Cancer Research

THE Committee on Growth of the National Research Council, acting for the American Cancer Society, is entertaining applications for grants and fellowships. Applications for new Grants in Cancer Research will be received until 1 October, 1949. Investigators now receiving grants will be notified individually regarding applications for the extension of these grants. Final decision on applications for the extension of these grants will be made in most cases soon after 1 February. Grants approved at this time ordinarily will become effective 1 July 1950.

Fellowship applications may be submitted at any time. Those received prior to 1 November will be acted upon by the Committee on Growth in December. Those received between 1 November and 1 March will be acted upon in April. Fellowships ordinarily will begin 1 July though this date may be varied at the request of the applicant.

During the past year the American Cancer Society, Inc., on recommendation of the Committee on Growth, has approved research grants and fellowships totaling over $2,000,000.

Communications regarding grants and fellowships should be addressed to Executive Secretary, Committee on Growth, National Research Council, 2101 Constitution Avenue, N.W., Washington 25, D. C.

Pregnancy Diagnosis Tests

(*Continued from page* 214)

The large mass of data have been handled in such a way that the reader can readily find the essential facts on the numerous pregnancy diagnosis tests. Furthermore, there is one index of these tests and a second index referring to the animals in which pregnancy is being tested.

The author claims to have presented his data as an aid for those who are interested in the veterinary aspect of pregnancy diagnosis. The reader will find that this book has a broader application. Gynecologists, obstetricians and investigators interested in reproduction may use this book as a handy source of information on the available data pertaining to the pregnancy tests in general.

Arthur M. Cloudman

We regret to announce that Dr. Cloudman died suddenly on August 8, 1949.

CONGENITAL INHERITANCE OF INGUINAL HERNIA

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Bethany College, W. Va.

IN a recent text book on genetics Colin[2] states that "comparatively few abnormalities of the muscular system are known to exist, and only in a few cases have they been shown to be mendelian." The author goes on to say that "rupture is a defect in which heredity undoubtedly plays a part." The main forms of hernia, which involve the rupture of the abdominal wall, are inguinal, femoral, abdominal and umbilical. According to Iason,[5] approximately twenty-five percent of individuals afflicted with hernia give a family history of it in parents and grandparents.

According to Macready[6] fathers transmit the defect more often to sons than to daughters and mothers transmit it more often to daughters than to sons. Inguinal hernia is most usual in males and the femoral and umbilical types appear most often in females. Moorehead[8] concludes that in every case of inguinal hernia there is a congenital weakness in the fascia at one or both ends of the inguinal canal. Bauer, Fischer, and Lenz,[1] from their studies, concluded that hernia is inherited and suggest that it is dominant. However, they cite no pedigrees. Watson [10] reported Macready as saying, "I recall a patient with inguinal hernia associated with a partially descended testis who said that both his father and his paternal grandfather had the same condition." West[11] cites several reports on the inheritance of hernia in man and in laboratory mammals and livestock. He further describes and discusses two pedigrees showing a rather frequent occurrence of hernia through three to five generations. The first pedigree was a study of "a family given largely to laborious pursuits," but the second "includes for the most part individuals of sedentary occupation, many of them engaged in professional life." The frequency of hernia in both groups is about the same and "tends to indicate that inherited conditions frequently manifest themselves regardless of occupation or environment." These studies record the histories of some cases of congenital hernia but the herniation was induced in most cases in the first study by excessive physical exertion. In the second study there was no excessive physical exertion. However, in both pedigrees there were few instances of the inheritance of congenital hernia. These data apparently indicate that a "weakness of the abdominal wall was inherited, probably as a dominant." Additional evidence for the inheritance of hernia has been furnished by the study of identical twins where often both members of a pair have ruptures. Roberts[9] suggests that hernia is a sex-linked dominant with variable expression. Evans,[3] after the study of a recent pedigree of hernia which included one son with hernia (probably inguinal), concludes that "The method of inheritance, although apparently dominant, cannot be regarded as settled and may prove to be different for different forms of hernia." Montagu[7] reports a study covering two generations. The father had inguinal hernia and so did two of his three sons. Two of the three sons were monozygous twins. However, of the pair of monozygotic twins only one twin was afflicted. Montagu[7] concluded that the family history "suggests a sex-linked dominant gene."

Inheritance

The data reported to the writer apparently indicate a clear case of the inheritance of congenital inguinal hernia. The study covers five generations. How-

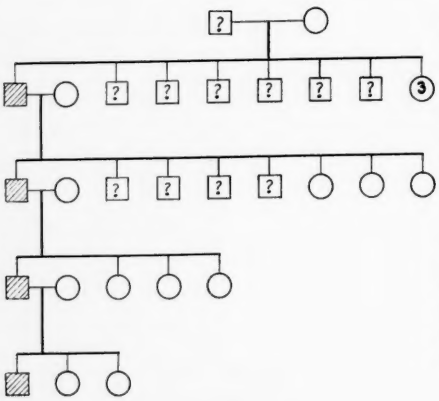

Figure 8

For four generations there has been a clear record in this family of father-to-son inheritance of inguinal hernia. Since this characteristic is for anatomical reasons far more frequent in the male sex, its inheritance as a sex influence character, dominant in males may have an anatomical rather than a physiological basis.

ever, there are no reliable data available concerning the parents of the first generation reported. In this family there were ten children, seven of whom were boys and three were girls. Of these children, reliable information is available on only one of the sons from whom the later generations described in this paper, are descended. This son had an inguinal hernia from birth. Unfortunately, there is no record as to whether this was a single or a double inguinal hernia. See Figure 8.

The male just mentioned was the father of eight children—five boys and three girls. Of these sons there is indubitable evidence that one had a double inguinal hernia from birth. Members of the family who are now living report that other brothers were afflicted with hernia but they can give no information as to the nature of the defect.

Of this generation the one son, i.e., the one concerning whom we have definite information, was the father of a family of four children, one boy and three girls. The son, who furnished the data for this report, had a congenital double inguinal hernia. The herniation was much more pronounced on the right side than on the left. This was true also of the man's father.

The man mentioned above has a family of three children, one boy and two girls. The boy was born with a double inguinal hernia. However, "his left side only showed the weakness of the muscles and did appear only as a swollen place at times." See Figure 8.

Conclusions

The data presented show a condition of congenital double inguinal hernia in the males of three generations and in one male of the fourth generation, the generation farthest removed in point of time from the present. From the evidence presented, the conclusion seems plausible that double inguinal hernia is inherited and that we are here dealing with a sex influenced gene, dominant in males. There is some indication that in the family studied, the defect is gradually becoming less prominent on the left side of the body.

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ARE THE POMES AMPHIDIPLOIDS?

A Note on the Possible Origin of the Pomoideae

HAIG DERMEN*

THE breeding behavior of commercial varieties of apples has proved to be more involved than that of many diploid plants, which suggests that the apples are complex polyploids. In 1930 Darlington and Moffett[1] and later Moffett[3,4,5] made certain cytological observations from which they concluded that the basic chromosome set of the Pomoideae, $x = 17$, is derived from the ancestral seven-chromosome genom of the Rosaceae. Vaarama[12] has recently offered data in support of this hypothesis. He reports that in the tetraploid apple variety Hibernal: "Tetravalents are most commonly found. Tri- and pentavalent associations are somewhat rarer. Hexavalents appear in somewhat greater number than bivalents." Moffett[4] states for some diploid forms in Pomoideae that "in the great majority of divisions examined, 17 bivalents were observed, though in a small percentage multivalent associations were found." He also reports higher valency than tri- and tetravalency in triploids and tetraploids, respectively. Darlington and Moffett have put great emphasis on "secondary pairing" of chromosomes in the diploids, forming "in extreme cases, seven groups" at meiosis, as evidence of Pomoideae being derived from a simpler ancestral form. Derivation from such an *ancestral form* would imply autopolyploidy. Such was also Sax's[6] interpretation of the Darlington and Moffett hypothesis. However, such an implication has been denied by Moffett in a later publication.[5]

Sax[8] has suggested:

> In diploid genera of the Pomoideae there is frequently considerable "secondary pairing" of chromosomes, suggesting remote homologies within the basic genom of 17 chromosomes.... The Pomoideae are allopolyploids, derived from remote ancestral types with 8 and 9 chromosomes, which in turn may have been derived from species or genera with 7 pairs of chromosomes.

Sax[7] failed to find any evidence of multivalent chromosome association in diploids. Multivalency found by Moffett and Vaarama either may be due to the "remote homology" suggested by Sax or it may be in part due to segmental interchanges which may have occurred between some chromosomes.

Moffett in his later publication[5] seems to agree with Sax's idea, as he remarks:

> It seems probable therefore that even if the Pomoideae have arisen from crosses between genera having basic numbers of 8 and 9 [as suggested by Sax] these basic numbers are themselves derived from the primary seven, and merely form a step in the evolution of the group.

Amphidiploid origin of the Pomoideae, as suggested by Sax,[8] seems more likely to be the correct interpretation. In support of this possibility, a case in *Lactuca* may be mentioned here. In *Lactuca*, as in Rosaceae,[6] there are species with $x=8$, $x=9$, and $x=17$ chromosomes. Thompson[9] and Thompson, Haney, and Kosar[11] have presented evidence that $x=17$ forms of *Lactuca* are of amphidiploid origin, resulting from the cross $x=9$ by $x=8$. The hybrids with the somatic chromosome number 17 were sterile.[10] By the colchicine method, Thompson and Kosar (personal communication) have succeeded in obtaining partially fertile amphidiploid plants, with $2x=34$ chromosomes.

The history of the origin of Pomoideae may be a parallel case to that in *Lactuca*.

*Cytologist, Division of Fruit and Vegetable Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, U. S. Department of Agriculture.

Assuming that Pomoideae resulted from intergeneric crosses between species with $x=8$ and $x=9$, an immediate hybrid plant, either completely or partially self-sterile, with the somatic chromosome number 17 would be the result. Because of its unbalanced meiotic condition, such a plant would not reproduce itself by a normal sexual method and would exist for the duration of its life only. The occasional union of fertile unreduced gametes would produce amphidiploid plants with a balanced meiotic condition. Such amphidiploid plants would be expected to reproduce themselves and they might only remotely resemble the parents of the initial hybrid. The existing diversity between various genera in the Pomoideae suggests that each genus is the result of hybridization between certain species of perhaps two genera. This phylogeny is quite different from the concept that all the genera of the Pomoideae have been "derived from a simple ancestral type, and that mutation and minor changes in chromosome morphology have been responsible for generic and species differentiation" as had been suggested earlier by Sax.[7]

Neither autopolyploid plants in other genera, such as we can now induce by the colchicine method, nor aneuploid plants which can be produced from autotriploid plants, differ radically from the diploid forms.[2] In many cases they are highly sterile and their sterility becomes greater as their degree of polyploidy is increased. If Pomoideae had been derived merely by autopolyploidy and aneuploidy it would be reasonable to expect that some species in Pomoideae would be found with basic numbers of 7, 8, or 9, or exact multiples of these. None with such numbers has been recorded thus far, and perhaps none may be found, for the reasons advanced above. Only a mixture of diverse characters of species of two or more distinct genera could account for the great taxonomic differences between the characters in the Pomoideae and those of other forms of Rosaceae. A careful taxonomic study of Rosaceae with the amphidiploid origin of Pomoideae kept in mind may possibly suggest what genera of Rosaceae would be the most likely ancestors of Pomoideae. With such clues, it may not be entirely impossible to reconstruct anew some of the existing forms.

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SEED PRODUCTION IN HORSERADISH

W. W. WEBER*

HORSERADISH is an extremely hardy perennial plant. It forms a rosette of erect, long-petioled leaves. The leaf blade is tough, long, dark green, and has crenate, sinuate, or pinnate margins. The plant may or may not form a flower stalk depending on environmental conditions. The inflorescence is a terminal or axillary raceme of short-pedicelled, small white flowers (Figure 9). Horseradish is highly sterile: the anthers are usually abortive, and normally no pods or seeds are formed, although seedless pods have been reported.

Taxonomically, horseradish has been placed in various genera by different botanists. It is known as, *Nasturtium armoracia* Fries, *Roripa armoracia* Hitchc., *Radicula armoracia* Robinson, *Cochlearia armoracia* L., *Armoracia armoracia* Britton, and *Armoracia rusticana* Gaertn. The latter taxonomic designation is preferred by most workers. *Cochlearia macrocarpa* var. *hungarica* is considered by Engler as a primitive form of horseradish, but other workers agree that this is the ordinary plant which has escaped from cultivation. Brzezinski[1] believes that horseradish is not a natural species but a hybrid.

Horseradish is grown throughout the northern United States. The largest commercial acreage is found in Missouri and Illinois, although considerable horseradish is grown in Michigan, Ohio, Pennsylvania, northern New Jersey, Connecticut, Massachusetts, and Washington. Horseradish is usually cultivated as an annual crop. It produces a fleshy, white taproot which is used as a condiment. The thickened lateral roots are generally used as sets for propagation.

Two types of horseradish are available to the commercial grower. These are known as the "common" and the Bohemian varieties. The "common" horseradish is a broad-leaved, hollow-petioled type and may have originated in eastern Europe. Nearly all of our commercial acreage is planted with this variety. It produces a root crop of good quality in spite of being very susceptible to white rust and horseradish mosaic diseases. It is reported by Pound[5] to be approximately 100 percent virus infected. The Bohemian horseradish, also known as the Maliner Kren or Bayersdorf strain, is a smooth, narrow-leaved, solid-petioled variety and is believed to have originated in southern Europe. It lacks root quality and yield but appears to be highly field resistant to both the white rust and horseradish mosaic diseases. The disease resistance of the Bohemian variety makes it a valuable type to use in a breeding program for horseradish improvement.

Seed Production

A number of clones of horseradish were collected from different areas in Wisconsin and planted in the greenhouse during the fall and winter of 1947 and 1948. They came from commercial plantings and from uncultivated areas where the plants had persisted as weeds for many years. The collection of clones contained a fair sampling of both the "common" and the Bohemian varieties. These original selections proved very fortunate, because one clone of "common," though completely pollen-sterile, produced ovaries in which about 5 percent of the ovules contained normal-appearing gametophytes; and two clones of Bohemian matured some functional pollen. Subsequent collections during

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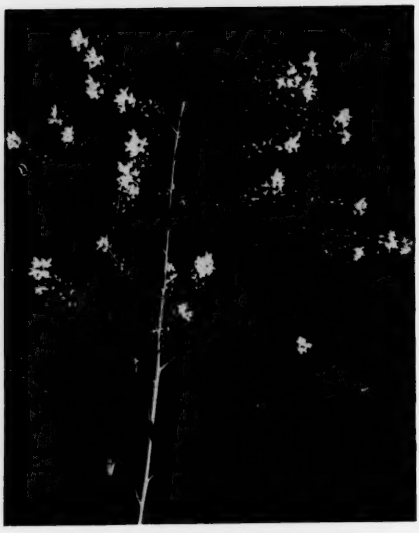
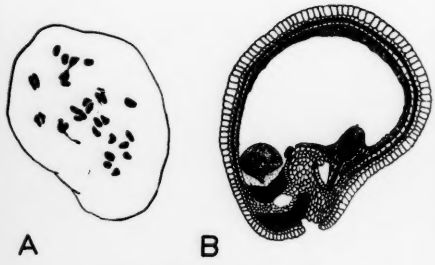


INFLORESCENCE OF BOHEMIAN HORSERADISH

Figure 9

The inflorescence is a terminal or axillary racene of short-pedicelled, small white flowers. Horseradish seldom blooms under natural conditions. It can be induced to blossom profusely under greenhouse conditions. The photograph is approximately 1/6 natural size.



SEED DEVELOPMENT

Figure 10

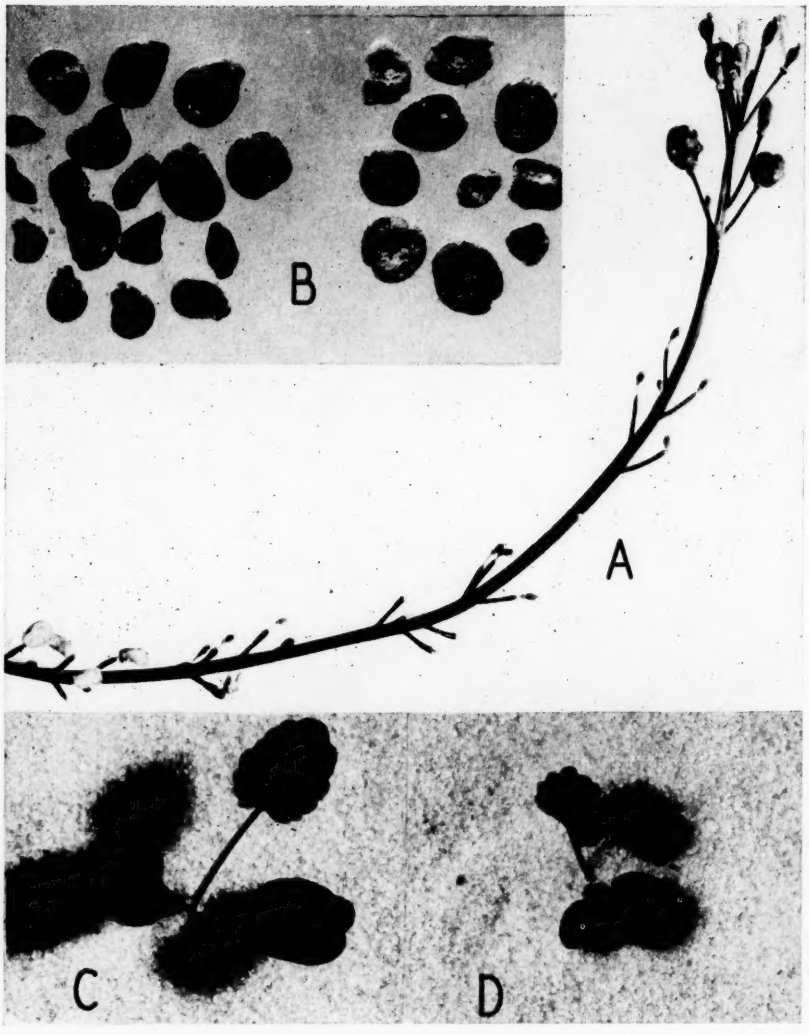
Viable seed is rarely produced on horseradish. *A*—Diakinesis showing partial pairing of the chromosomes and other irregularities. *B*—Longitudinal section of developing seed. Seed development appears to be normal if functional pollination and fertilization has occurred.

the spring and summer of 1948 resulted for the most part in completely sterile clones.

No clones of "common" thus far examined have produced any functional pollen. The pollen grains show signs of abortion in the early stages of development. Two clones of Bohemian produced some functional pollen. In one about 25 percent, and in the other about 50 percent of the grains were normal in appearance.

It is possible to effect fruit development on several of the pollen sterile "common" clones by applying pollen from one of the pollen producing Bohemian clones to the receptive stigmas. Fruits develop very rarely following selfing of the pollen producing clones. This is surprising since 30-40 percent of the ovules contain normal appearing female gametophytes. A higher percentage of fruit development would be expected unless the phenomenon of self-incompatibility found in the *Brassicas* by Kakizaki,[3] Detjen,[2] and Pearson,[4] also applies to horseradish. The fruits develop rapidly for a short period following pollination, then development stops in a high percentage of the cases, with usually only a few continuing to grow. The developing fruits (Figure 11, *A*) are more or less distorted in appearance. Usually only one seed develops within a fruit, with two seeds rarely being present. The fruits mature in three to four weeks, at which time they dehisce, leaving the seed attached to the placenta wall or permitting it to drop. Dehiscence usually occurs while the fruits are still green in color. It is necessary to keep close watch of the maturing pods, if one wishes to collect the few seeds that develop.

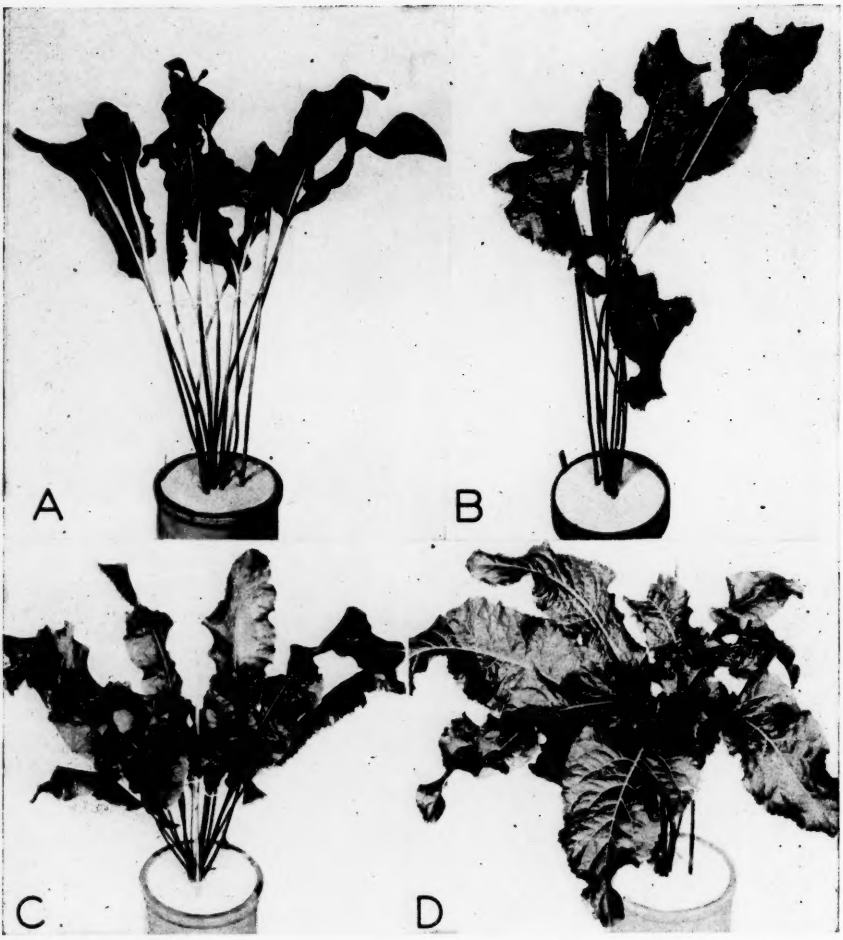
The cross between the two types of horseradish proves to be the most effective means of securing seed. The seed harvested varied from fairly well developed to badly shrunken kernels (Figure 11, *B*). The selfed seeds are smaller and usually not as well developed as those resulting from the cross. Generally the mature kernels are brown to gray in color. Many of the well developed seeds are viable at maturity but lose the ca-



FRUITS, SEEDS AND SEEDLINGS

Figure 11

Fruits, seeds and seedlings resulting from a cross between the "common" and Bohemian varieties of horseradish. *A*—Fruiting branch showing distorted fruits, aborting fruits and naked ovaries which had not been stimulated to develop. *B*—Seeds collected from ripened fruits. They ranged from fairly plump to badly shrunken specimens. *C*—Normal horseradish seedling. *D*—Seedling with fused cotyledons. The fruits and seedlings are approximately natural size. The seed is enlarged about six times.



VARIABILITY OF HYBRIDS

Figure 12

Hybrid horseradish plants are highly variable and vigorous. *A*—Selfed offspring from the pollen producing Bohemian clone. It has smooth, narrow leaves; long, solid petioles and it appears to be resistant to white rust and horseradish mosaic. *B, C* and *D* are hybrids between the "common" and Bohemian varieties of horseradish. *B*—A plant with ruffled, broad leaves and long, partly hollow petioles. *C*—A plant with smooth, broad leaves and short, hollow petioles. *D*—A plant with ruffled, very broad leaves and short hollow petioles. Plants *B, C* and *D* are susceptibe to white rust and appear to be resistant to horseradish mosaic.

pacity to germinate after short periods of storage.

Seedlings

A few seeds were obtained from the small percentage of pollinated flowers that produced fruits. Some of these seeds were planted. Irregularities noticed in the seedling stage, such as fused cotyledons (Figure 11, *D*) are similar to those common among *Brassica* seedlings. Twelve offspring are being grown. Eleven of these plants represent crosses between the "common" and the Bohemian types of horseradish. The twelfth plant is an offspring of the pollen producing clone (Figure 12, *A*). These plants vary widely in morphological characters as shown in Figure 12 *B*, *C*, and *D*. The selfed offspring has petioles with solid piths while the crosses have hollow petioles. The leaf blades of the various plants differ in size, shape, smoothness, and color. The offspring are vigorous and show no evidence of the presence of virus. Preliminary tests indicate that the F_1 plants, of the cross between the white rust and virus-susceptible "common" clone and the resistant Bohemian clones, are segregating for these characters.

Cytological Studies

The somatic number of chromosomes as determined by aceto-carmine root tip smears is 32 in both the "common" and Bohemian varieties of horseradish. Irregularities occur in the premeiotic and meiotic divisions in microsporogenesis. Partial pairing of the chromosome is to be seen at diakinesis (Figure10, *A*). There are numerous lagging chromosomes on the meiotic spindles. These spores with the requisite number of chromosomes develop into pollen grains. The gametes are formed in the pollen grains prior to dehiscence of the anthers.

The ovary contains 16 to 20 ovules. Meiosis is likewise irregular in megasporogenesis. Oftentimes the spore fails to continue growth or the gametophyte starts to develop and breaks down prior to maturity so that only a small percentage of the ovules contain female gametophytes that appear normal. There is the occasional accumulation of food materials in the nucellar regions of nonfunctional ovules following pollination.

Elimination of the virus from suitable types through the production of seed should increase commercial yields. The production of fertile lines of horseradish through cross pollination and selection would greatly stimulate improvement work with this plant. Fertile lines would furnish material for a study of the inheritance of disease resistance. It would also furnish material to investigate the status of horseradish as a natural species and its taxonomic position in the scheme of classification.

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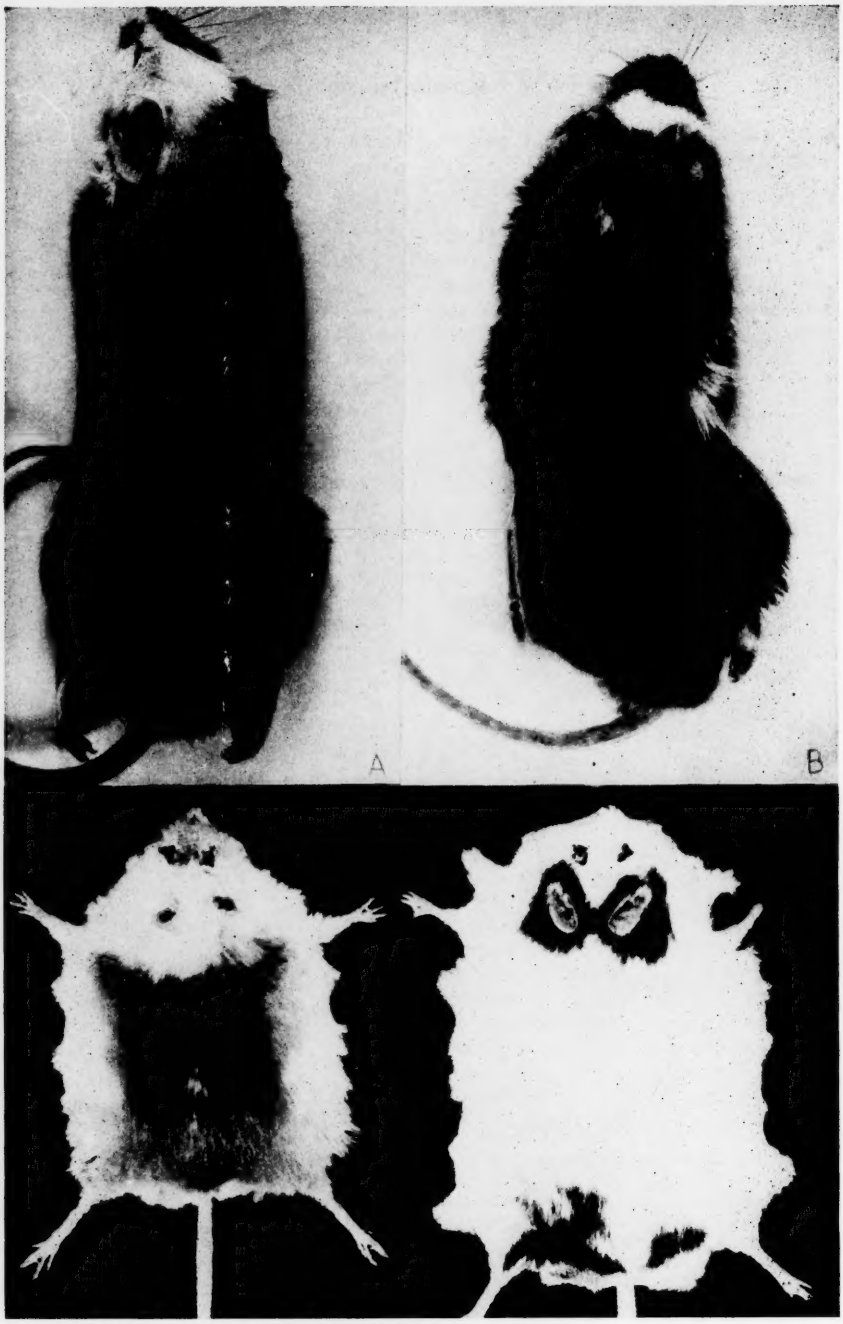

VARIED PIEBALD PATTERNS

Figure 13

A—An s^1s^1 mouse with white spotting limited to the head, and showing only slight asymmetry. *B*—Asymmetrical pattern of s^1s^1, with the ventral white area extending up on right side, and a small white spot over the shoulders. *C*—Extreme expression of s^1s^1, with the ventral area extending almost to the hind legs. A few white hairs appear in the dorsal mid-line. *D*—A typical piebald homozygous for the piebald-complex, showing the distribution of color around ears and base of tail.

A NEW RECESSIVE SPOTTING GENE

In the House Mouse*

TERRY PULLIG

IN 1944 a number of self hairless males of *Mus musculus* were being rotated every four days in the pens containing the spotted F_1 females of a cross between self hairless males and dominant spotted females in order to determine the crossover value between *hr* and *W* according to Gates and Pullig.[2] The hairless males used in this experiment had been carefully checked for self by inbreeding for six and seven generations and they had shown no evidence of any type of spotting. For months the classes of crossover and non-crossover individuals in the experiment remained relatively constant for any consecutive 100 individuals. Then suddenly these classes became most irregular. Due to these irregularities the experiment was discontinued and the self hairless males were put into breeding pens with piebald females. In order to check each male they were not rotated. One of these breeding pens showed the following results:

Date	*No. in litter*	*Self*	*Spotted*
2/6/45	5	4	1
[Killed]			
5/27/45	11	5	6(all ♀ ♀)
[Killed]			
6/2/45	8 (3 dead)	2	3
[Killed]			
6/9/45	9	0	9(2 ♀ ♀ and 7 ♂ ♂)
[Saved 1 ♂ and 2 ♀ ♀]			

The relative numbers of self and piebald indicate clearly that the hairless male was heterozygous for a type of piebald allelic to the common type (*ss*). However, the type of spotting differed noticeably from the generally recognized type, particularly with respect to the region around the head.

The spotted individuals had in general the new type of spotting pattern on the head and the ventral side. This white area appeared as a band around the ears extending in varying degrees down the ventral side and looking much like a white scarf tied around the head with free ends of various lengths.

According to Dunn and Charles,[1] the amount of white and the location of the spots are extremely variable in piebald mice, but there is evidence of a general pattern for these white areas. The areas include the feet and tail, which are white in nearly all spotted forms; the belly spot; the nose-tip and the blaze between the eyes, which when confluent, forms the "white face"; paired shoulder spots which often join to form a collar; paired lumbar spots which often join to form a belt. In the lighter forms more of these areas are white, and the individual white spots are greater in extent. The rump and areas about the eyes, ears, and cheeks are nearly always colored (Figure 13 *D*).

From the litter saved, brother-sister matings were made and have been continued for the most part for the last four years. Due to the fact that the first individuals were heterozygous for the piebald-complex, these brother-sister matings produced some offspring that tended toward the recognized piebald type of spotting (*ss*). Rigid selection has tended to eliminate the old piebald factor. Only those males and females were saved that had the fewest spots on the body and a band on the head. After a few generations of selection it was found that a few pens tended to produce less of the piebald type of spotting; so all others were then discarded. From these few pens the present stock has been derived. At the present it breeds fairly true often having only a few or no white hairs on the body (Figure 13 *A, B, C*). Sometimes the white on the ventral side extends up one side (Figure 13*B*). The band around the head is rather sym-

*Contribution No. 92, Department of Zoology, Physiology, and Entomology, Louisiana State University.

metrical but there are some with asymmetrical bands (Figures 13 *A* and 13 *B*). There have been a few with the entire head and part of the neck white (Figure 13 *C*). On one mouse the entire anterior half of the body was white. The amount of white on the ventral side varies from a little on the throat only, to almost all the ventral part (Figure 13 *C*).

This new spotting complex has been tested in small numbers only with the Bagg Albino, the snowy belly, and the silver because these carry no spotting genes. In no case was there any evidence of spotting in the offspring of these crosses; thus indicating that this new spotting complex is completely recessive. In a later paper the F_2 and backcross results as well as linkage percentage will be reported. In crosses between the common piebald type and many of the selfs there is almost always some spotting in the F_1 while this new gene complex fails to express any evidence of its presence in the heterozygous condition.

Conclusions

1. In this one self male a gene of the self complex has mutated to a recessive spotting gene which may be known as s^1.
2. This new spotting gene complex is completely recessive.

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✦

Microtechniques for the Study of the Chemical Morphology and Physiology of Cells

THIS is the first comprehensive book* that has been published in the particular field that it covers. As such it sets a high standard of excellence for any others that might be attempted. It is marked by a clarity of presentation and description that is not usually encountered in most manuals.

Micromethods of the types described in this work are being used more and more extensively by biologists in various fields of work, so that even a less well written and organized manual would prove very valuable and useful. The first part (162 pages) of the book deals with methods of microscopically identifying and localizing various chemical constituents in sectioned tissues and cells. Lison's *Histochemie Animale*, published in 1936, was the last adequate treatment of this subject. The present book not only brings this material up to date but includes many newer quantitative techniques and procedures such as Caspersson's, and Engström's methods of ultraviolet and Roentgen absorption histospectroscopy, Scott and Packer's analytical electron microscopy and methods of radioautography. The second part of the book (274 pages) deals with microchemical techniques. Here the work of Linderstrom-Lang and Holter (to whom the book is dedicated and in whose laboratory the author worked for a time) appropriately figures very largely. A very short third part (8 pages) presents an adaptation to micro scale of the now well-known bioassay techniques with microorganisms. The fourth part of the book (28 pages) describes methods for the mechanical separation of various components of cells. It includes methods for the isolation of nuclei, chromatin threads, mitochondria, chloroplasts, and other structures or particles. The bibliography (35 pages) is quite extensive. A useful list of manufacturers is appended and the text contains frequent reference to individual firms where special apparatus or material may be obtained.

For cytogeneticists as well as other workers in the general field of cellular physiology this work should prove extremely useful. It is evident, however, from the rate of progress in this field that frequent revision will be required. This is well illustrated by the fact that the author considered it essential to add to the bibliography an appendix of 55 papers that appeared in the nine months subsequent to completion of the manuscript.

ALBERT TYLER
California Institute of Technology

*Techniques of Histo- and Cytochemistry by David Glick; published by Interscience Publishers, Inc., New York City, 531 pp., $8.00; 1949.